**Report No:** DOT/FAA/AM-00/34

**Title and Subtitle:** A fatality caused by hydrogen sulfide produced from an accidental transfer of sodium hydrosulfide into a tank containing iron sulfate and sulfuric acid.

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**Abstract:** The National Transportation Safety Board has an agreement with the Federal Aviation Administration (FAA) that the FAA's Civil Aerospace Medical Institute (CAMI) provide toxicological services for selected surface transportation accidents. Under this agreement, postmortem biosamples from a hazardous chemical accident fatality were submitted to CAMI for toxicological evaluation. The victim succumbed from breathing the hydrogen sulfide (H₂S) gas produced by an accidental transfer of sodium hydrogen sulfide (NaHS) from a tanker truck to a tank containing 4% sulfuric acid (H₂SO₄) and iron(II) sulfate (FeSO₄). After inhaling the gas, the 55-year old male Caucasian truck driver was dead at the scene. Autopsy examination of the decedent's body revealed pulmonary edema and passive congestion in lungs, spleen, kidneys, and adrenal glands. The submitted samples were analyzed for carbon monoxide, cyanide, alcohols, and drugs. Since a potential exposure to H₂S was involved, blood was also analyzed for sulfide (S²⁻). The analysis entailed isolating S²⁻ from blood as H₂S using 0.5 M H₃PO₄, trapping the gas in 0.1 M NaOH, and determining the electromotive force using a sulfide ion specific electrode. Carbon monoxide, cyanide, or ethanol was not detected in blood, but acetaminophen at a therapeutic concentration of 14.3 µg/mL of blood was found, and metoprolol was detected in the blood, liver, and kidney samples. Analysis further revealed the presence of S²⁻ in blood at the level of 1.68 µg/mL. This S²⁻ concentration is approximately 2 times higher than that reported in the blood of 2 separate fatalities associated with accidental exposures to H₂S. The blood S²⁻ value in the present case was about 34 times higher than the blood S²⁻ concentration (< 0.05 µg/mL) in normal subjects. The observed pulmonary edema and the passive congestion in various organs were also in agreement with the pathological characteristics of H₂S poisoning. Since H₂S toxicity manifests rapidly by inhibiting the cytochrome oxidase system, causing histotoxic cellular hypoxia, death occurs quickly. Based on the case history, pathological findings, and blood S²⁻ concentration, it is concluded that the cause of death was H₂S poisoning associated with a hazardous material accident in an industrial situation.

**Key Words:** Forensic Sciences, Toxicology, Acetaminophen, Metoprolol, Hydrogen Sulfide, Hazardous Material, Accident Investigation

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